1: Acta Pharmacol Toxicol (Copenh). 1981 Nov;49(5):334-53.

Inhibition of adrenergic neuroeffector transmission in rabbit pulmonary artery and aorta by adenosine and adenine nucleotides.

Husted S, Nedergaard OA.

The effect of adenosine and adenine nucleotides on sympathetic neuroeffector transmission in the rabbit isolated pulmonary artery and aorta was studied. Adenosine (10(-5)-3 x 10(-4)M) decreased the contractile response of pulmonary artery and aorta evoked by electrical-field stimulation. The decrease was reversible. No tachyphylaxis developed. Inhibition of either adenosine deaminase by deoxycoformycin (3.6 x 10(-6) M) or of adenosine transport by dilazep (3 x 10(-6)M) did not alter the inhibitory effect of adenosine on the neurogenic contractions in the pulmonary artery. However, deoxycoformycin plus dilazep markedly enhanced the inhibitory effect of adenosine. The calcium antagonists nifedipine (1.5 x 10(-8)M) and nimodipine (1.3 x 10(-8)M) had no effect on the adenosine-induced inhibition. This was also the case with theophylline (5 x 10(-5)M), atropine (10(-7)M), and the prostaglandin synthetase inhibitors indomethacin (5 x 10(-5)M and suprofen (3 x 10(-5)M). The contractile response of the pulmonary artery elicited by exogenous (-)-noradrenaline (NA; 10(-9)-3 x 10(-4)M) was essentially not altered by adenosine (10(-5)-3 x 10(-4)M). Adenosine (10(-4)M) did not alter the spontaneous 3H-outflow from rabbit aorta preloaded with 3H-(-)-noradrenaline (3H-NA). Adenosine (10(-5)-3 x 10(-4)M), ADP (10(-4)M), ATP (10(-5)M), and inosine (10(-4)M) diminished the overflow of tritium from pulmonary artery and aorta preloaded with 3H-NA. The spontaneous outflow of tritium from aorta preloaded with 3H-NA consisted of 3H-NA (17%), 3H-dihydroxyphenylglycol (3H-DOPEG; 30%), 3H-dihydroxymandelic acid (3H-DOMA, 4%), 3H-O-methylated and deaminated metabolites (3H-OMDA; 42%), and 3H-normethanephrine (3H-NMN; 2%). Adenosine (10(-5) and 10(-4)M) enhanced 3H-DOPEG and 2H-NMN, decreased 3H-NA, and did not alter 3H-DOMA and 3H-OMDA. The stimulation-evoked overflow of tritium for aorta preloaded with 3H-NA consisted of 3H-NA (31%), 3H-DOPEG (18%), 3H-DOMA (2%), 3H-ONDA (46%), and 3H-NMN (3%). Adenosine (10(-5) and 10(-4)M) enhanced 3H-NA and 3H-DOPEG, decreased 3H-OMDA and did not alter

3H-DOMA and 3H-NMN. Adeosine (10(-6)-10(-4)M) did not alter the accumulation of 3H-NA (10(-8)M) by aorta. It is concluded that adenosine inhibits vascular sympathetic neuroeffector transmission by diminishing the release of transmitter from the nerve terminals.

PMID: 6285668 [PubMed - indexed for MEDLINE]
